# In the United States Court of Federal Claims
**OFFICE OF SPECIAL MASTERS**
**No. 23-235V**

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| DEBBIE NEASE BOHANNON, *as mother of B.B.*, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent. | Chief Special Master Corcoran<br><br>Filed: January 2, 2025 |

*Lisa A. Roquemore*, Law Office of Lisa A. Roquemore, Rancho Santa Margarita, CA, for Petitioner.

*Mark K. Hellie*, U.S. Department of Justice, Washington, DC, for Respondent.

## ENTITLEMENT DECISION[1]

On February 16, 2023, Debbie Nease Bohannon, on behalf of her son, B.B., filed a petition seeking compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petition (ECF No. 1) ("Pet.") at 1. Petitioner alleges that B.B.'s May 14, 2021 receipt of a tetanus, diphtheria, and acellular pertussis ("Tdap") vaccine, "by itself or in concert with the [meningococcal conjugate] vaccine" also administered that same day, caused B.B. to develop type 1 diabetes mellitus ("T1DM"). Pet. at 4.

[1] Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Ruling will be available to the public in its present form. *Id.*

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) ("Vaccine Act" or "the Act"). Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

After an initial review of the filings, I informed the parties that the claim appeared unlikely to succeed, given the Program's consistent, twenty-plus year rejection of theories that T1DM could ever be vaccine-caused. But I afforded Petitioner the opportunity to demonstrate what *new* scientific/medical evidence or facts would support causation in this case – and to that end, both parties have filed briefs. Petitioner's Response to Order to Show Cause, dated April 29, 2024 (ECF No. 27) ("Br."); Respondent's Brief, dated May 30, 2024 (ECF No. 28) ("Opp."); Petitioner's Reply, dated June 17, 2024 (EC No. 29) ("Reply").

Now, having reviewed the parties' arguments along with the filed medical records, I deny compensation. The contention that *any* covered vaccines can cause T1DM *is not scientifically reliable*, as recognized in numerous prior well-reasoned decisions, nor has Petitioner offered an alternative, persuasive causation theory based on updated science applicable to diabetes or the vaccines B.B. received. And nothing about B.B.'s personal circumstances – including the fact that he received more than one vaccine at a particular time, within a year's timeframe, or experienced initial diabetes-related symptoms not long after vaccination – changes the analysis.

## I. Fact Summary

Because this case primarily turns on the "can cause" element of the test for entitlement, only an abbreviated review and summary of the medical record is required.

B.B. was born on March 23, 2009, and was thus 12 years old at the time of the relevant vaccinations. Ex. 1 at 32, 135. He has no medical history bearing on the case, and was current on his vaccinations as of the spring of 2021 (including receipt of the Tdap vaccine in the summer of 2020). *Id.* at 114. Although some family members had previously developed type 2 diabetes later in life, Petitioner alleges that no other family member had previously been diagnosed with T1DM. Br. at 3; Pet. at 2.

On May 14, 2021, B.B. received a physical examination at his pediatrician's office. Ex. 1 at 32-33, 35, 135-36. B.B. weighed 155 pounds and was noted to be gaining weight. *Id.* at 32, 135. At this time, he received the Tdap and meningococcal conjugate vaccines. *Id*. at 31-32, 136. B.B.'s urinalysis was normal, with no evidence of glucose or ketones. *Id*. at 135. There is no direct medical record evidence of any post-vaccination reaction for over a month thereafter. (However, Petitioner reported in later medical records that B.B. had experienced nausea on the evening of the vaccine. *See, e.g.*, *Id.* at 137 (November 2021 record).

On June 26, 2021 (now approximately six weeks post-vaccination), B.B. was taken to the emergency room with complaints of high blood sugar, vomiting, generalized weakness, and urinary frequency. Ex. 7 at 8. Petitioner informed treaters (consistent with witness statements offered in this case) that at an outing to Disneyland on May 28, 2021 (two weeks post-vaccination),

B.B. had become very tired and had vomited a couple of times. *Id*. Petitioner also alleged that she and a family friend noticed that B.B. started drinking water excessively at this time. Pet. at 1. Since then, B.B.'s symptoms had progressed. Ex. 7 at 8.

B.B.'s weight in the emergency room was now 141 pounds, significantly less than at his May 2021 physical. Ex. 7 at 9. Based upon presentation and Petitioner's history recollection, the treating doctor proposed diabetes and diabetic ketoacidosis as diagnostic explanations, and an insulin drip was initiated. *Id*. at 10. While in the emergency room, B.B.'s blood sugar decreased from over 600 mg/dL to 194 mg/dL (normal range 70-99 mg/dL). Ex. 7 at 12, 16. In addition, B.B.'s hemoglobin A1c[3] was 12.4% (normal range 4.8% to 5.6%). *Id*. at 16.

On June 27, 2021, B.B. was transferred to a pediatric ICU for further monitoring. Ex. 7 at 10-11, 66. He was discharged home the following day, with orders to follow up with an endocrinologist. *Id.* at 66. A few days later, B.B. was taken to see endocrinologist Gnanagurudasan Prakasam, M.D., for follow-up with his diabetes. Ex. 5 at 23. B.B.'s blood sugar level was now elevated at 339 mg/dL. *Id.* Dr. Prakasam discussed a variety of diabetes treatment and management issues and prescribed some insulin. *Id.* at 26.

For the rest of 2021, B.B. followed up with Dr. Prakasam for his diabetes. Ex. 5 at 16, 33, 53; Ex. 1 at 48-50. In 2022, B.B. (still in his "honeymoon" period[4]) was able to discontinue all insulin use. Ex. 5 at 40 This has continued into 2023. Ex. 37 at 5; Ex. 38 at 1, 3. B.B.'s last labs from June 2023 showed a hemoglobin A1c of 5.7, so only slightly elevated. Ex. 38 at 3.

## II. Expert Reports

### A. *Petitioner's Expert - Dr. David Axelrod*

Petitioner filed an expert report at the initiation of this matter, setting forth her causation theory. *See* Report, dated February 13, 2023, filed as Ex. 8 (ECF No. 7-1) ("Axelrod Rep.").

Dr. Axelrod graduated from the University of Michigan Medical School in 1974 (after obtaining his bachelor's degree at Michigan as well). Ex. 9, filed as ECF No. 7-2 ("Axelrod CV") at 1. He completed two residencies in internal medicine, one at the University of Toronto and one at William Beaumont Hospital, followed by additional residencies with a fellowship in allergy,

[3] The hemoglobin A1c test is a blood test that measures a person's average blood sugar over a three-month period. *Testing for Diabetes and Prediabetes: A1C*, CDC, https://www.cdc.gov/diabetes/diabetes-testing/prediabetes-a1c-test.html#cdc_testing_why_get_tested-what-does-the-a1c-test-measure (last visited Nov. 13, 2024).

[4] The "honeymoon phase" of T1DM is the period of time after the initial presentation of symptoms, while the body continues to produce enough insulin to lower initially-high blood glucose levels. *Understanding Type 1 Diabetes*, American Diabetes Association, https://diabetes.org/about-diabetes/type-1 (last visited Nov. 13, 2024).

immunology, and rheumatology at McGill University. Axelrod CV at 1. He then served as a fellow for the National Institutes of Health in the Clinical Immunology Laboratory. *Id.* Dr. Axelrod is board certified in medicine, allergy and immunology, adult rheumatology, and medical laboratory immunology. *Id.* He currently works in private practice, with the vast majority of his patients having allergies, immunologic conditions, or autoimmune rheumatic diseases. *Id.* Dr. Axelrod does not appear, however, to conduct research in immunologic matters relevant to the theory expressed in this case, nor is he an expert in the study or treatment of diabetes (let alone the interaction between it and the immune system).

Dr. Axelrod began with a summary of B.B.'s medical history and how it resulted in the T1DM diagnosis. Axelrod Rep. at 1-3, 5. He observed that T1DM is understood to reflect "an absolute deficiency of insulin secondary to the loss of pancreatic β cells," and listed the diagnostic criteria for the condition. *Id.* at 3; Leonard C. Harrison, "Type 1 Diabetes," in *Clinical Immunology: Principles and Practice* (R. Rich ed., 2019), filed as Ex. 1 (ECF No. 7-3) ("Harrison"). Dr. Axelrod particularly emphasized that T1DM has two subtypes, with Type 1A (the autoimmune form) confirmed by evidence of autoantibodies to a number of pancreatic islet cell antigens, although he also noted that the disease process is driven by specific T cells (rather than antibodies produced by B cells). Axelrod Rep. at 3-4. He also cited evidence that the external stimulation of certain viral infections, like Rotavirus, was thought to be a possible trigger, occurring due to molecular mimicry between viral protein components and amino acid sequences in pancreatic islet cells (although he referenced no literature specifically connecting any of the antigenic components of either putatively-causal vaccine at issue). *Id.* at 4, 6. Rather, he invoked the more general proposition that *some* autoimmune diseases could occur due to infection, via the mechanism of molecular mimicry (in which amino acid sequences in a protein, contained in a virus or bacterium, resemble a self-component, resulting in a mistaken cross-reaction). *Id.* at 6.

Dr. Axelrod next turned to his causation theory, which relied heavily on the notion that a vaccination's stimulation of the innate immune process would later lead to a cross-reactive autoimmune attack (as part of a secondary, adaptive immune response to vaccination). The presentation of foreign antigens to the immune system sparks an initial, innate reaction – manifesting in part by the production of cytokines that "induce oxidative stress and induce [] posttranslational modifications in β-cell proteins that may render them immunogenic." Axelrod Rep. at 4. Cytokine production could also, he maintained, activate certain self-reactive immune cells in susceptible individuals, overcoming the immune system's regulatory safeguards and causing or propagating autoimmune disease. *Id.* at 6. B.B.'s vaccination event caused a rise in cytokines (reflected in the GI symptoms he reported prior to his hospitalization event). *Id.* at 9. These in turn propagated damage to the relevant pancreatic cells responsible for insulin production. *Id.* Thereafter, Dr. Axelrod opined, the adaptive response in reaction to the vaccines caused the production of autoantibodies responsible for further harm. *Id.* Existing autoreactive T-cells were also likely activated, contributing to the disease process. *Id.*

To support the idea that vaccine-induced cytokines could initiate a disease process resulting in T1DM, Dr. Axelrod offered several items of literature. For example, he referenced a study that (he argued) established that certain proinflammatory cytokines could constitute "[t]he final death effector molecule" for pancreatic cell apoptosis. I. Chang et al., *Role of Calcium in Pancreatic Islet Cell Death by IFN-γ/TNF-α,* J. Immunol. 7008 (2004), filed as Ex. 11 (ECF No. 7-4) ("Chang"). Chang noted that apoptosis of pancreatic B cells was "the final step in the development of [T1DM]," and sought to identify the specific cytokines involved. Chang at 7008. But Chang's actual focus is on the extent to which *calcium ion concentrations* (relevant to cellular electrical activity and signaling) in the pancreatic beta cells would be impacted by synergies between different kinds of cytokines. *Id.* at 7009. Thus, although Chang does observe cytokines playing a role in the process leading to the destruction of these cells, it does not find that the cytokines *initiate* the process – let alone what would increase their presence in the pancreas sufficiently to become harmful, whether a vaccine or infection. A comparable article reached much of the same conclusions – that cytokines are involved in the final destruction of the pancreatic islet cells – but without elucidating anything more specific that would connect *vaccine*-produced cytokines to that destruction. *See* S. Kim et al., *Apoptosis of Human Islet Cells by Cytokines,* 12 Immune Network 3:113 (2012), filed as Ex. 12 (ECF No. 7-5) ("Kim").[5]

In addition, Dr. Axelrod noted that cytokine release could impact gastrointestinal symptoms like vomiting (perhaps in an attempt to associate B.B.'s reported initial symptoms with the vaccinations at issue). Axelrod Rep. at 5, 9; G. Goel et al., *Cytokine Release and Gastrointestinal Symptoms After Gluten Challenge in Celiac Disease,* 5 Sci. Adv. 1 (2019), filed as Ex. 14 (ECF No. 7-7) ("Goel"). Goel, however, does not involve T1DM but instead celiac disease, and found merely (via serologic testing) that "acute gluten exposure" (occurring when someone with celiac disease encountered gluten in food) resulted in rapid increases in cytokine elevations (occurring less than 10 hours after exposure). Goel at 1-2. While vomiting was a common associated adverse event that Goel's authors deemed "a clinical proxy for cytokine levels," Goel says nothing about comparable symptoms in the progression of diabetes – and the speed at which the cytokine-associated symptoms manifested is not comparable to the weeks that passed before B.B. experienced GI-associated symptoms. *Id.* at 3.

Another article (also referenced by Dr. Axelrod in prior cases) purports to show how vaccination can quickly result in the production of a variety of pro-inflammatory cytokines.

[5] Dr. Axelrod also tried to analogize pancreatic islet cell dysfunction to the context of a kind of arthritis, invoking an animal study in support. *See* H. Jin et al., *IL-6 Promotes Islet β-Cell Dysfunction in Rat Collagen-Induced Arthritis,* J. Diabetes Rs. 1 (2016), filed as Ex. 13 (ECF No. 7-6). But this article not only involves a distinguishable disease process (rheumatoid arthritis ("RA")) – which *itself* is not understood to be triggered by an infection or vaccine (despite its autoimmune character) – but involves exploration of a possible association between RA and *type 2 diabetes* (not the version at issue), as well as the degree to which chronic inflammation in RA could "complicate" the diabetes's progression. Thus (and yet again), Dr. Axelrod attempted to leverage an article *looking at* cytokine involvement in an unrelated autoimmune-mediated disease to reach conclusions in the disparate context of T1DM and vaccine causation.

Axelrod Rep. at 5-6; Kashiwagi et al., *Production of Inflammatory Cytokines in Response to Diphtheria–pertussis-tetanus (DPT), Haemophilus Influenzae Type B (Hib), and 7–valent Pneumococcal (PCV7) Vaccines*, 10 Human Vaccines & Immunotherapeutics 677, 678 (2014), filed as Ex. 15 (ECF No. 7-8) ("Kashiwagi"). Kashiwagi was an *in vitro* study comparing the levels of inflammatory cytokines in the sera of 61 vaccine recipients with febrile illness, against 18 recipients without febrile illness, 24 hours after vaccination (a fairly short period of time). Kashiwagi at 677. The study's authors began with peripheral blood mononuclear cell cultures and then introduced different combinations (separately or concurrently) of the DTaP, Hib, and/or PCV7 vaccines in order to determine the levels of cytokine production in the cell cultures. *Id.;* Axelrod Rep. at 6.

Relying on Kashiwagi, Dr. Axelrod maintained that the relevant proinflammatory cytokines implicated in his theory are produced beginning six hours after vaccination and continue to increase until 24 hours following vaccination. Axelrod Rep. at 6. These elevated levels were found to persist after the 24-hour post-vaccination period. *Id.* Yet there are sound reasons to distinguish Kashiwagi from the present matter, and to find that its conclusions are less compelling than Dr. Axelrod proposes. For example, the results showing increased cytokine production were mainly seen in connection with a form of the pneumococcal vaccine, which is not relevant to this case. Kashiwagi at 679. More significantly, Kashiwagi found no real difference between the two compared serum groups, beyond the fact that one particular cytokine was elevated in individuals experiencing a febrile illness. *Id.* at 680. Because Kashiwagi's authors admitted that "[v]accine-specific innate inflammatory responses…have not been sufficiently investigated regarding cytokine production using difference vaccines," they could not characterize this difference as significant (*id.* at 678), and ultimately concluded that more analysis was required. *Id.* at 683.

Dr. Axelrod next attempted to demonstrate how molecular mimicry could have played a mechanistic role in the vaccination-T1DM association he alleged. Axelrod Rep. at 7-9. He set forth the contents of the two vaccines at issue in this case, and their specific antigenic character. *Id.* at 7. He then proposed "target antigens" for T1DM, based on his prior contentions as well as evidence of what autoantibodies B.B. had possessed. *Id.* Because he could show homologic sequence equivalence between vaccination antigenic components and self-protein components relevant to T1DM, Dr. Axelrod deemed the possibility of a molecular mimicry-driven autoimmune process preponderantly established.

Connecting this aspect of the proposed causation theory to B.B.'s actual presentation hinged on the results of certain testing. In late June 2021, B.B. had tested positive for the presence of a particular autoantibody considered a T1DM biomarker: glutamic acid decarboxylase ("$GAD_{65}$") autoantibodies.[6] Ex. 6 at 3; Axelrod Rep. at 2, 4. And there has been at least some

[6] GAD is an enzyme utilized by the pancreas to produce Gamma-aminobutyric acid (GABA), a neurotransmitter. The presence of anti-GAD antibodies is a predisposing factor for the development of diabetes. *What is Glutamic Acid*

scientific speculation that certain infectious processes (such as a rotavirus infection leading to GI symptoms) might cause the production of $GAD_{65}$ autoantibodies due to mimicry between components of the virus and islet cells. Harrison at 963. Because, Dr. Axelrod reasoned, amino acid sequence homology could be demonstrated between $GAD_{65}$ and components of the Tdap vaccine, it was likely the vaccine could cause the production of these autoantibodies. Axelrod Rep. at 7-8.

Besides contending the vaccines B.B. received could cause his T1DM, Dr. Axelrod proposed that the timeframe in which diabetes symptoms manifested was medically acceptable, when measured from the date of vaccination. Axelrod Rep. at 10-11. He referenced again Kashiwagi, which established a relatively fast (within a day) upregulation of cytokines after vaccination. *Id.* at 10. This timeframe was consistent with when B.B. first felt gastrointestinal distress (albeit two weeks later from the medical records). Then, the adaptive process in reaction to antigenic presentation could peak within two weeks. *Id.* And the fact B.B. had received a prior Tdap vaccination the year before suggested a faster memory response in 2021, when exposed to the same antigens again. *Id.* at 11.

B. *Respondent's Experts*

1. Dr. Steven Willi - Dr. Willi, a pediatric endocrinologist, offered one written report in this matter. Report, dated January 20, 2024, filed as Ex. B (ECF No. 25-1) ("Willi Rep."). Dr. Willi rejects the contention that any of the vaccines B.B. received could have caused his T1DM.

Dr. Willi is a Professor of Pediatrics at the University of Pennsylvania. Willi CV, dated February 21, 2024 (ECF No. 25-31) ("Willi CV"). He works as an attending physician at the Children's Hospital of Philadelphia and is the director of the Diabetes Center for Children. Willi CV at 1. Dr. Willi received his M.D. from Johns Hopkins School of Medicine, and completed his pediatric residency and Endocrinology and Diabetes fellowship at the Children's Hospital of Philadelphia. *Id.* He is board-certified in Pediatrics and Pediatric Endocrinology. *Id.* at 2. In his role as Director of the Diabetes Center for Children, Dr. Willi oversees the care of over 2,500 children with T1DM. Willi Rep. at 2. He has been published over 180 times and has received numerous research grants from NIH, CDC and the FDA, as well as from industry and private foundations. *Id.*

Dr. Willi began his report with a summary of B.B.'s medical history. Willi Rep. at 2-4. He agreed the record evidence supported B.B.'s diagnosis of T1DM – specifically type "1a" diabetes, which meant it was the form that was autoimmune-mediated, with a cross-reaction against insulin-

*Decarboxylase (GAD)?*, Very Well Health, https://www.verywellhealth.com/glutamic-acid-decarboxylase-6890566 (last visited Dec. 4, 2024).

producing "beta cells," or cells of the pancreatic islets, resulting in insulin deficiency. *Id.* at 4. He noted that this version takes months to unfold, and it is "often years before clinical disease becomes evident." *Id;* M. Atkinson et al., *Type 1 Diabetes,* 383 Lancet 69, 70, 72-73 (2014), filed as Ex. B, Tab 2 (ECF No. 25-3) ("Atkinson"). Indeed, due to the long lag phase between the development of the destructive autoantibodies and diabetes symptoms manifestation, treatments are often aimed at *slowing* the progression from the occurrence of destruction of the beta cells to when symptoms clinically manifest. Willi Rep. at 4.

Dr. Willi noted that the question of whether environmental stimuli could trigger T1DM in a genetically-susceptible individual (although he did not include vaccines as such a factor) had previously been considered by the medical/scientific community. Willi Rep. at 4. In particular, researchers more than 50 years ago speculated that viral infections might be causal. *Id.* at 5; D. Gamble, *Seasonal Incidence of Diabetes Mellitus,* 3 Brit. Med. J. 631, 633 (1969), filed as Ex. B, Tab 9 (ECF No. 25-10) ("Gamble") (abrupt onset of diabetes might reflect infection followed by autoimmune response). However, subsequent research demonstrated the unreliability, if not falsehood, of such putative associations. K. Coppieters et al., *Virus Infections in Type 1 Diabetes,* 2 Cold Spring Harb. Perspect. Med. 1, 2 (2012), filed as Ex. B, Tab 10 (ECF No. 25-11) ("Coppieters") (although "a variety of viruses have come under scrutiny" for causing T1DM, "[i]n the majority of cases . . . associations are weak, irreproducible, or were in some instances convincingly disproved"). At most, some research indirectly suggested an association with a single viral infection – enterovirus (not the flu virus) – but the results could not be corroborated by more methodologically-sound studies. Coppetiers at 2-3; K. Vehik et al., *Prospective Virome Analyses in Young Children at Increased Genetic Risk for Type 1 Diabetes,* 25 Nat. Med. 1865, 1870-71 (2019), filed as Ex. B, Tab 11 (ECF No. 25-12) (prolonged enterovirus infection somewhat associated with pancreatic islet cells autoimmune processes).[7] And there has even been some evidence that certain infections might play a *protective* role against the development of T1DM. *See, e.g.*, Coppetiers at 10 (discussing findings relating to coxsackie virus infections).

In contrast, Dr. Willi contended, vaccines have been credibly and fairly-conclusively shown *not* to be associated with T1DM, resulting in a scientific community consensus against the existence any such relationship. Willi Rep. at 5-6. In support, Dr. Willi referenced several large-scale epidemiologic studies. *See generally* A. Beyerlein et al., *Vaccinations in Early Life are not Associated with Development of Islet Autoimmunity in Type 1 Diabetes High-Risk Children: Results from Prospective Cohort Data*, 35 Vaccine 1735 (2017), filed as Ex. B, Tab 18 (ECF No. 25-19) ("Beyerlein"); E. Morgan et al., *Vaccinations and Childhood Type 1 Diabetes Mellitus: A*

[7] Dr. Willi also sought to rebut a purported association between the COVID-19 infection and T1DM, referencing his own research into the subject. *See generally* Willi Rep. at 5. But that infection is not comparable to the flu virus, nor (and more importantly) is the COVID vaccine *itself* comparable to the inactivated flu vaccine, and therefore these contentions merit no further discussion.

*Meta-Analysis of Observational Studies*, 59 Diabetologia 237 (2016), filed as Ex. B, Tab 19 (ECF No. 25-20) ("Morgan").

Beyerlein combined data from two prospective cohort studies involving German children deemed at high risk for T1DM (based on a familial history of the disease), ultimately resulting in a sample of more than 1900 subjects. Beyerlein at 1736. Vaccination records for the cohort allowed the study's authors to consider a large number of vaccines routinely administered to children – including both vaccines at issue in this case. *Id.* at 1736, 1738-39. As its authors explained, diabetes is typically preceded by a preclinical period of islet autoimmunity. *Id.* at 1735. The objective of this study was to determine whether vaccinations were associated with an increased risk of islet autoimmunity. *Id.* Beyerlein's authors ultimately concluded that "early vaccinations are not associated with either increased or reduced risk" for T1DM. *Id.* at 1740.

Morgan, a meta-analysis,[8] combined the data considered in 23 individual studies involving 16 vaccines, including vaccines containing the three Tdap components. Morgan at 237, 241. The studies compared vaccination rates in children who subsequently developed T1DM and in control children. *Id.* at 237. It found no association between routine childhood vaccinations and T1DM. *Id.* at 242.

Given the reliability of these kind of scientific studies and findings, Dr. Willi stressed, "such venerable bodies as the Institute of Medicine"[9] have embraced the view that childhood vaccines do not likely cause T1DM. Willi Rep. at 7; *Adverse Effects of Vaccines: Evidence and Causality* 571 (K. Stratton et al., eds., 2012), filed as Ex. D. Tab 16 (ECF No. 25-48) (the "IOM Report") (based on review of five epidemiologic studies, the IOM Report expressed "a high degree of confidence" for a "null association" between the Tdap vaccine and T1DM; no conclusions were expressed, however, with respect to the meningococcal vaccine).

Dr. Willi overall deemed Dr. Axelrod's opinion to be unpersuasive (especially since Dr. Axelrod's expertise as an adult rheumatologist made him less likely to have any background in treating pediatric T1DM). For example, he maintained that Dr. Axelrod described *type 1b* diabetes in his theory – not the subtype B.B. had been diagnosed with, and not otherwise corroborated by the medical record. Willi Rep. at 6. He also noted that Dr. Axelrod proposed cytokines could cause beta cell death as reflected in experimental studies, but that (putting aside there was no medical record evidence B.B. had sufficient levels of comparable proinflammatory cytokines to be dangerous) these kinds of studies had not been replicated in human testing – and that it was

[8] A "meta-analysis" is a quantitative statistical analysis of several separate but similar studies. It is conducted in order to test the pooled data for statistical significance. *Meta-analysis*, Merriam-Webster Online, https://www.merriam-webster.com/dictionary/meta-analysis (last visited Nov. 13, 2024). While a meta-analysis is not necessarily more persuasive simply because it aggregates the findings of smaller studies, it is considered to have a valid methodology.

[9] The Institute of Medicine is now known as the National Academy of Medicine.

unreliable to extrapolate their conclusions to humans. *Id.* at 6-7. At the same time, other diabetes-specific studies revealed that blocking certain kinds of cytokines did *not* delay pathogenic progression – undermining the conclusion of the centrality of these cytokines in the autoimmune process causing the destruction of these pancreatic islet cells. *See generally*, A. Moran et al., *Interleukin-1 Antagonism in Type 1 Diabetes of Recent Onset: Two Multicentre, Randomised, Double-Blind, Placebo-Controlled Trials*, 381 Lancet 1 (2013), filed as Ex. B, Tab 22 (ECF No. 25-23) ("Moran") (blockade of Interleukin-1 was found to be ineffective in delaying the progression of type 1 diabetes); C.J. Greenbaum et al., *Receptor Blockade Does not Slow β Cell Loss in New-Onset Type 1 Diabetes*, 6 JCI Insight 1 (2021), filed as Ex. B, Tab 23 (ECF No. 25-24) ("Greenbaum")[10] (blockade of Interleukin-6 was found to be ineffective in delaying the progression of type 1 diabetes).

Dr. Willi also challenged Dr. Axelrod's reliance on molecular mimicry as a mechanism explaining how vaccines could trigger T1DM. Willi Rep. at 7. He noted no evidence at all, from a medical/scientific standpoint, that associated T1DM with a vaccine-induced cross-reaction mechanism – although in support, he cited additional research specific to other vaccines. *See, e.g.*, H. Viskari H et al., *Live Attenuated Enterovirus Vaccine (OPV) is not Associated With Islet Autoimmunity in Children With Genetic Susceptibility to Type 1 Diabetes: Prospective Cohort Study*, 61 Diabetologia 203 (2018), filed as Ex. B, Tab 24 (ECF No. 25-25) (finding that the live enterovirus vaccination is not associated with the development of Type 1 diabetes); M. Rogers et al., *Lower Incidence Rate of Type 1 Diabetes after Receipt of the Rotavirus Vaccine in the United States, 2001-2017*, 9 Sci. Rep. 7727 (2019), filed as Ex. B, Tab 26 (ECF No. 25-27).

The timeframe in which B.B.'s T1DM's symptoms first manifested post-vaccination was also not, in Dr. Willi's estimation, medically acceptable enough to suggest vaccine causation was likely. Willi Rep. at 7. The record revealed B.B. first likely experienced such symptoms within *two weeks* of his May 2021 vaccinations. *Id.* But a number of studies established that T1DM could take months to years before manifesting clinically. *Id.* (citing R.A. Insel et al., *Staging Presymptomatic Type 1 Diabetes: A Scientific Statement of JDRF, the Endrocrine Society, and the American Diabetes Association*, 38 Diabetes Care 1964, 1965 (2015), filed as Ex. B, Tab 3 (ECF No. 25-4) ("[i]n children and adults, the rate of progression from onset of b-cell autoimmunity to glucose intolerance and then to symptomatic disease is variable, lasting from months to decades")).

2. Dr. You-Wen He, M.D., Ph.D. – Dr. He, a medical doctor and academic immunologist, also prepared a written report for Respondent. *See* Report, dated November 30, 2023, filed as Ex. D (ECF No. 25-32) ("He Rep.").

Dr. He is a Professor of Integrative Immunobiology in the Department of Integrative Immunobiology at Duke University School of Medicine. He CV, dated February 21, 2024, filed

[10] Dr. Willi co-authored Greenbaum.

as Ex. E (ECF No. 25-61) ("He CV"). He received his medical degree from the Fourth Military Medical University in China and received his PhD from the Miami School of Medicine. He CV at 1. Dr. He went on to complete a senior fellowship in the Department of Immunology at the University of Washington and completed his residency at Qindu Hospital in China. *Id.* Dr. He has been conducting research in immunology since he graduated from medical school in 1986. He Rep. at 1. Over the past 27 years, he has been invited to lecture nationally and internationally on the topic of host immune responses to microbial infections and tumors. *Id.* Dr. He has also served as a co-Principal Investigator for four clinical trials focusing on cancer immunotherapy using personalized cancer vaccines. *Id.* Finally, he has been published extensively and has served as an ad hoc reviewer for more than 30 scientific journals. *Id.*; He CV at 7-18.

Dr. He's report included a summary of the medical record, as well as a recapitulation of the nature of T1DM and B.B.'s diagnosis, but he ultimately deferred to Dr. Willi on such matters (and did not otherwise dispute how either side characterized the alleged injury or propriety of the diagnosis). He Rep. at 2-3, 5-7. Dr. He also allowed that certain specific environmental factors have been reasonably associated with diabetes, such as perinatal factors as well as enteroviral infections. *Id.* at 3, 7.[11] But Dr. He strongly disputed that the Tdap vaccine – or any vaccine for that matter – could cause T1DM.

Dr. He's challenge to Petitioner's causation theory relied on several points, beginning with a review of its individual components. Dr. Axelrod first maintained the initial innate response to vaccination would produce proinflammatory cytokines that would create the background circumstances necessary for destruction of pancreatic islet cells. He Rep. at 8-9. But the contention that cytokine production due to vaccination could have the same destructive potential as a wild infection was, in Dr. He's estimation, incorrect, and assumed the impact of the two immune triggers was the same. *Id.* at 9. In fact, the human immune response was keyed to respond far more robustly to a wild infection – in comparison to a vaccine, administered in a peripheral muscle (which would inherently limit infectious progression, in contrast to the "natural routes" followed by an infection beginning in the nasal or respiratory passages of the body). *Id.* at 9-11.

In addition, Dr. He maintained that literature offered to establish a vaccine-cytokine upregulation association had less reach than Dr. Axelrod proposed. One article offered by Dr. Axelrod on this point, Kim, involved *in vitro* studies showing the destructive potential of cytokines in the context of pancreatic cells. But to obtain experimentally-meaningful results, Kim's authors

[11] B.B.'s reported vomiting, which Dr. Axelrod suggested was proof of a cytokine reaction, could just as well have been evidence that B.B. was at that time experiencing an enterovirus infection, Dr. He maintained, given the record evidence of pre-vaccination illness (then thought to possibly reflect COVID-19). He Rep. at 7. Dr. He also later proposed that this reaction could simply reflect post-vaccination malaise. *Id.* at 19. Ultimately, the record evidence is too equivocal on the nature of B.B.'s vomiting episode – and even if it could be deemed an initial T1DM symptom, its timing (relatively close to vaccination) plus the greater lack of preponderant proof of vaccine causation at all, means that it does not assist Petitioner's causation contentions.

induced the production of levels of cytokines that were inordinately and unnaturally high – nowhere near what would be expected to occur after vaccination. He Rep. at 16-17; Kim at 2. Kashiwagi, by contrast, showed far lower levels of cytokines after vaccination that would be necessary to be pathologic. Kashiwagi at 4-5. It was thus unlikely, Dr. He maintained, that vaccination could ever promote an initial pathogenic process simply due to its expected stimulation of the production of cytokines.

The next sub-component of Dr. Axelrod's theory involved the contention that the adaptive immune response would feature molecular mimicry between the vaccine antigenic components and self-tissues/structures specific to the relevant pancreatic cells, leading to progression of disease process through an autoimmune attack. He Rep. at 12-14. But Dr. He argued that molecular mimicry between amino acid chain components of antigens and self-molecules is extremely common in nature but does not inerrantly cause disease. *Id.* at 15; B. Trost et al., *Bacterial Peptides are Intensively Present Throughout the Human Proteome*, 1 Self Nonself 71, 72 (2010), filed as Ex. D, Tab 23 (ECF No. 25-55) ("Trost"). Thus, the shared peptide sequences Dr. Axelrod had observed in his report between components of the Tdap vaccine and self-structures were not meaningful (with Trost specifically noting commonalities specific to bacteria that the Tdap vaccine was aimed at thwarting). Trost at 72.

Finally, Dr. He disputed that the follow-on immune responses that Dr. Axelrod proposed could further the attack were likely to occur. For example, Dr. Axelrod maintained that "self-reactive lymphocytes" already present in the human body could further encourage an autoimmune process. He Rep. at 11. But such cross-reactive immune cells were not only common, but were likely only to encourage *existing* infectious and autoimmune processes, as opposed to act as primary causes. *Id.* Thus, this aspect of the immune response could not be *causal* of an initial pathogenic process, and it had not otherwise been shown by Dr. Axelrod that the vaccines at issue could "break tolerance," i.e. disrupt the immune regulatory process that kept these potentially cross-reactive immune cells in check. *Id.* at 11-12. The same was true, Dr. He proposed, for contentions about "bystander activation," in which non-specific immune cells would propagate autoimmune damage. *Id.* at 12. This process required some initial antigenic attack, such as an infection, to occur, which Dr. He maintained was not comparable to vaccination. And in fact, recent studies had demonstrated that vaccine-induced bystander activation was a beneficial and expected part of the immune reaction, as opposed to a pathologic occasion. *Id.*; F. Horns et al., *Memory B Cell Activation, Broad Anti-influenza Antibodies, and Bystander Activation Revealed by Single-Cell Transcriptomics*, 30 Cell Rep 905, 911 (2020), filed as Ex. D, Tab 21 (ECF No. 25-53).

Besides challenging the persuasiveness and reliability of Dr. Axelrod's theory, Dr. He (echoing points made Dr. Willi) noted the extensive epidemiologic evidence undercutting the proposition that any childhood vaccines can cause T1DM. He Rep. at 3-4; *See e.g.*, Beyerlein;

Morgan; A. Hviid et al., *Childhood Vaccination and Type 1 Diabetes*, 350 New Eng. J. of Med., 1398, 1403 (2004), filed as Ex. D, Tab 3 (ECF No. 25-35) ("Hviid") ("there appears to be no support for any causal relation between childhood vaccination and type 1diabetes").

Other studies suggested a familial history associated with diabetes made T1DM more likely – and that applied specifically to B.B. He Rep. at 18-19; *See e.g.*, G. Dahlquist et al., *The Swedish Childhood Diabetes Study—Results From a Nine Year Case Register and a One Year Case-Referent Study Indicating that Type 1 (Insulin-Dependent) Diabetes Mellitus is Associated With Both Type 2 (non-insulin-dependent) Diabetes Mellitus and Autoimmune Disorders*, 32 Diabetologia 2 (1989), filed as Ex. D, Tab 26 (ECF No. 25-58); S Sipetic et al., *Family History and Risk of Type 1 Diabetes Mellitus*, 39 Acta Diabetol 111 (2002), filed as Ex. D, Tab 27 (ECF No. 25-59); M.B. Weires et al., *Familiality of Diabetes Mellitus*, 115 Exp. Clin. Endocrinol Diabetes 634 (2007), filed as Ex. D, Tab 28 (ECF No. 25-60).

## III. Parties' Contentions

*Petitioner*

Petitioner maintains the claim is viable - although her brief makes little mention of Dr. Axelrod's theory (other than noting he had in fact *provided* an opinion). Br. at 19. Thus, Petitioner's response to my Order to Show Cause includes no discussion of how Dr. Axelrod's contentions about cytokine production could trigger T1DM. She also does not cite any recently-published medical or scientific literature involving vaccine causation and T1DM. The only "evidence" she cites is a list of VAERS case reports. *See* Br. at 9, 46-32. And while she acknowledges that a great deal of existing medical literature discusses how vaccines do *not* cause T1DM, she argues that such literature "*do[es] not say that they cannot in rare cases*." Br. at 7.

Petitioner then makes a generalized statement on molecular mimicry, claiming that the theory continues to find acceptance in the medical community as a reasonable mechanism for some autoimmune injuries (but without explaining why it has any credibility in the context of T1DM).[12] Br. at 7. Petitioner also attempts to highlight some facts specific to B.B.'s medical history that she contends differentiate this case from prior matters. For example, she observes that B.B. received a Tdap vaccine on July 29, 2020, and another dose "10 months later" in May 2021, later suggesting that this temporal sequence was unusual or increased the chance of an adverse reaction. Br. at 2, 6 ("[t]his Tdap was administered way too early"); *see also* Reply at 2 (referencing Centers for

[12] Other possible avenues Petitioner proposes as worthy of consideration (even if Dr. Axelrod has not yet proposed them) are facially irrelevant. For example, Petitioner expresses the desire to "explore the CRM197" conjugate in the pneumococcal vaccine as a basis for causation, adding that some Program decisions have deemed this vaccine capable of causing Guillain-Barré syndrome ("GBS"), an autoimmune peripheral neuropathy. Br. at 7. Of course, not only is GBS not the injury at issue – but the pneumococcal vaccine *is also not one of the vaccines at issue* in this case.

Disease Control publications advising against over-frequent administration of tetanus-containing vaccines – albeit due to concerns for immediate reaction as opposed to development of diabetes). She also emphasizes that, as of the date of the May 2021 vaccination, B.B.'s urine testing revealed no glucose or ketones – but then repeat testing did so on June 26th, after he began to display other symptoms consistent with diabetes. *Id.* at 3, 8. She contends that this short timeframe is somehow unusual. *Id.* at 8. And the receipt of the meningococcal vaccine at the same time as the Tdap vaccine in May 2021 is also deemed a risk factor, with Petitioner invoking the meningococcal package insert as support. *Id.* at 6; Reply at 2.

Second, Petitioner endeavors to distinguish many of the prior cases (discussed in greater detail below) where a theory of vaccine causation leading to T1DM was advanced but rejected. *See generally* Br. at 9-15. Some are inapplicable, she contends, because the petitioners alleged significant aggravation of their *pre-existing* diabetes. *Id.* at 9, 13; *Hennessey v. Sec'y of Health & Hum. Servs.*, No. 01-190V, 2009 WL 1709053 (Fed. Cl. Spec. Mstr. May 29, 2009), *mot. for review den'd*, Fed. Cl. 126 (2010); *E.S. v. Sec'y of Health & Hum. Servs.*, No. 17-480V, 2020 WL 9076620 (Fed. Cl. Spec. Mstr. Nov. 13, 2020), *mot. for review den'd*, 154 Fed. Cl. 149 (2021). In others, the injured vaccinees developed diabetes almost two years after their last vaccination, a longer timeframe than what is at issue here. *Id.* at 14; *Baker v. Sec'y of Health & Hum. Servs.*, No. 99-653V, 2003 WL 22416622 (Fed. Cl. Spec. Mstr. Sept. 26, 2003); *Meyers v. Sec'y of Health & Hum. Servs.*, No. 04-1771V, 2006 WL 1593947 (Fed. Cl. Spec. Mstr. May 22, 2006).

Petitioner also cites a case not involving T1DM at all, but the distinguishable injury of autoimmune limbic encephalitis ("ALE") – likely because that matter involved the same two vaccines at issue in this case. *Agarwal v. Sec'y of Health & Hum. Servs.,* No. 16-191V, 2020 WL 5651683 (Fed. Cl. Spec. Mstr. Aug. 31, 2020) (Tdap and meningococcal vaccines). The injured party therein developed ALE, which is associated with GAD antibodies. Br. at 15. On June 27, 2021, B.B. was also found to possess antibodies to GAD. Ex. 6 at 3. The relevant child in *Agarwal*, however, had experienced a fever within three days of vaccination and was taken to the emergency room shortly thereafter, suffering demonstrable and alarming symptoms far sooner, and of a different nature, than what B.B. experienced herein. *Agarwal*, 2020 WL 5651683, at *3-7. Petitioner nevertheless deems *Agarwal* supportive of her theory because it has been shown that the relevant autoantibody is also possessed by patients with T1DM (although it has not been demonstrated at all that a vaccine can cause this autoantibody, nor has it been established that the presence of GAD antibodies establish T1DM susceptibility). Br. at 15-19.[13] In support, Petitioner

[13] Although Petitioner's detailed discussion of *Agarwal* references testimony and evidence offered in that case that has not been filed herein, I do not fault this per se, since I also have invoked numerous prior cases, and "what's sauce for the goose is sauce for the gander." However, *Agarwal* would be immensely more useful as guidance *if* it involved the relevant injury – for not all autoimmune injuries are the same, and cannot presumed to be for purposes of determining Program causation. And it remains uncontestable: Petitioner cannot identify a single Program case in the entirety of the history of the Program where T1DM was ever found to be caused by *any* vaccine, regardless of *Agarwal*'s outcome.

offers an extensive recapitulation of the testimony and evidence from that case, concluding that because the same two vaccines "led to an autoimmune event" in *Agarwal*, the same was possible here – albeit for an entirely different kind of illness. *Id.* at 19.

In addition, Petitioner proposes that to reject her claim at this stage would be unfair and/or constitute procedural error. She maintains the Program "is not about bright line rules," but is remedial, and therefore she deserves the chance to offer further expert support for her claim. Br. at 19-20. She claims she cannot bulwark her theory otherwise, and that the claim's novel theory should be permitted to be explored. *Id.* at 20-21. Thus, she requests the chance to obtain an endocrinologist (to explain the variance in glucose testing between the day of vaccination and later). *Id.* at 21. She will otherwise be deprived of a full and fair opportunity to explore her theory. *Id.* at 22.

*Respondent*

Respondent seeks dismissal of the claim, arguing that Petitioner's causation theory is unreliable and was insufficiently-substantiated by Dr. Axelrod and the independent evidence he relies upon. The aspect of his theory invoking molecular mimicry, Respondent contends, has been persuasively addressed, and rejected, in prior decisions. Opp. at 10, 11-12. There was also little evidence offered showing any vaccine-T1DM association, in comparison to reasoned and reliable studies undermining that conclusion. *Id.* at 10-11, *citing* Beyerlein at 1; Morgan at 1; and the IOM Report at 588. And the contention was conclusory and unexplained to some extent as well. *Id.* at 11.

Further, the aspect of Dr. Axelrod's theory specific to the role of cytokines was similarly unreliable. Dr. Willi noted research establishing that simply blocking the production of cytokines did not curtail T1DM's progress. Opp. at 11 (*citing* Greenbaum at 2, Moran at 10). And the idea that vaccines could produce cytokines at levels akin to a wild infection was insufficiently substantiated. He Rep. at 11.

The impact of B.B.'s receipt of two Tdap vaccines plus a meningococcal vaccine in less than a year was also not likely to produce T1DM. Other cases rejecting vaccine causation in this context had also involved multiple vaccines received in a particular timeframe. *See, e.g.*, *Hennessey*, 2009 WL 1709053, at *6-7 (two hepatitis B vaccines received within 70 days of alleged T1DM onset). Petitioner had simply not shown any new scientific/medical developments that would suggest causation was any more likely than when the Office of Special Masters had considered the issue over the prior 20 years.

## IV. Procedural History

This case was filed in the winter of 2023. Notably (and before the matter had even been activated and assigned to a special master), Petitioner's initial filings *included* Dr. Axelrod's report – underscoring the extent to which Petitioner had prepared in advance to attempt to show how T1DM could be vaccine-caused. *See generally* ECF No. 7 (February 20, 2023 filings). The case was originally assigned to a different special master, and in September 2023, Respondent offered his Rule 4(c) Report contesting entitlement (ECF No. 18). The case was reassigned to me in February 2024,[14] and shortly thereafter (and in accordance with the orders set by the prior special master who had presided over the claim), Respondent filed expert reports from Drs. Willi and He. I subsequently issued my Show Cause Order, the matter was briefed by both sides, and it is now ripe for resolution.

## V. Applicable Legal Standards

### A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[15] There is no Table injury for T1DM, so Petitioner can only proceed herein with a causation-in-fact claim.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's

[14] Petitioner's counsel has also suggested that reassignment of this matter had sinister undertones. *See* Br. at 2 ("[i]t is difficult to discern whether this reassignment was a 'random' assignment"). In fact, the reassignment was merely the product of the fact that Special Master Oler (who initially presided over the matter) was then anticipating departure from OSM due to her pending nomination to serve as a judge for the Superior Court of the District of Columbia.

[15] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. App'x. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health and Hum. Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

Each *Althen* prong requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245 ("[p]lausibility . . . in many cases *may* be enough to satisfy *Althen* prong one" (emphasis in original)).

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Kalajdzic v. Sec'y of Health &*

*Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592 F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also Howard v. Sec'y of Health & Hum. Servs*., 2023 WL 4117370, at *4 (Fed. Cl. May 18, 2023) ("[t]he standard has been preponderance for nearly four decades"), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024) (unpublished). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V,

2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs.*, No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs.*, 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs.*, No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making

a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

*Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion

"connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

E. *Disposition of Case on Written Record*

I am resolving Petitioner's claim on the filed record. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The determination to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs*., No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs*., 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

## ANALYSIS

B.B.'s T1DM diagnosis is not in dispute, so the only matter to resolve is whether the Tdap vaccine or meningococcal vaccines – together or in concert – "can cause" T1DM.[16] A few points about the nature of T1DM (and accepted by both sides) warrant emphasis, for purposes of analysis.

T1DM is best understood as a progressive, metabolic syndrome, and one that occurs in part due to a "background of genetic susceptibility." Harrison at 959; Atkinson at 70. While environmental factors (including some viral infection) may also play a role in its progression, T1DM is not considered to be a disease directly attributable to some external, singular "hit" – like a viral infection – "given the long presymptomatic stage of the disease," with infections instead proposed *possibly* to hasten symptoms manifestation via a "nonspecific inflammatory insult." Harrison at 963. But only one infectious agent, enterovirus, has even been associated with T1DM – and the literature is uniform in noting that "there is no scientific evidence that any form of vaccination itself triggers" T1DM. *Id.; see also* Atkinson at 69 ("no specific agents with an unequivocal influence on pathogenesis have been identified").

### I. The Vaccine Program Has Consistently Rejected Claims that Vaccines Cause Diabetes

The Program has been repeatedly tasked with determining if T1DM could be vaccine-caused. *Such claims have never succeeded* –not a single time. *See, e.g.*, *E.S.*, 2020 WL 9076620 (finding that Petitioner did not establish that the HPV vaccine itself could worsen T1DM); *Crutchfield v. Sec'y of Health & Hum. Servs*., No. 09-0039V, 2014 WL 1665227 (Fed. Cl. Spec. Mstr. Apr. 7, 2014) (MMR vaccine not shown to be causal of T1DM), *mot. for review den'd*, 125 Fed. Cl. 251 (2014); *Hennessey*, 2009 WL 1709053 (finding that petitioner had "failed to establish any logical connection between his hepatitis B vaccinations and his T1D [type 1 diabetes]" and finding that his deteriorated condition was caused by "the natural progression of insulin dependence, rather than his vaccines"); *Meyers*, 2006 WL 1593947 (finding that the Petitioner failed to establish a causal link between DTP vaccine and type 1 diabetes); *Baker*, 2003 WL 22416622 (rejecting claim that multiple vaccinations caused type 1 diabetes).

[16] I am solely resolving the claim on the basis of Petitioner's inability to preponderantly establish the first *Althen* prong. Because Program claimants must satisfy all three prongs to be entitled to damages, the failure to establish any single prong is grounds for a claim's dismissal – and therefore no analysis of Petitioner's success in meeting the other two prongs is required. *Dobrydnev v. Sec'y of Health & Hum. Servs.,* 566 Fed. Appx. 976, 980 (Fed. Cir. 2014). I note, however, that even if Petitioner could satisfy the first prong, it is unlikely she could also establish the third. Existing science on T1DM establishes that it often has a lengthy and subclinical course before symptoms manifest – allowing for the likelihood that B.B.'s T1DM had already begun by the time of his vaccinations. And it has not otherwise been shown that either vaccine he received could trigger preexisting but subclinical T1DM.

The reasoning employed by special masters in reaching these determinations is instructive – in particular the older decisions, which predate this matter by up to 20 years. In *Baker*, for example, a petitioner alleged that her infant son's receipt of a series of childhood vaccinations at a six-month wellness visit resulted in his diagnosis of T1DM at the age of three. *Baker,* 2003 WL 22416622, at *1. But the special master firmly rejected the causation theory offered – which arose specifically from articles and research performed by the expert (Dr. John Classen) who testified for the petitioner. *Id.* at *28-30. The theory advanced therein, as here, involved the contention that islet cell function could be harmed by vaccination, although the manner in which this was alleged to occur was different than what is proposed in this case:

> In very young children, over two months of age, vaccination may trigger a process where the macrophages damage islet cells in the pancreas. In older individuals, some autoimmune process is probably ongoing, and the incubation period can be shorter because the person is already substantially damaged. . .The size of the individual matters. A small person does not need so many islet cells. . .In young persons, there would be a three-year interval to clinical disease. . .The factors involved in IDDM are genetic, and preexisting, as well as based on the individual's size. Everyone is not born with the same number of islet cells.

*Id.* at *2. The special master rejected the theory, however, noting that (as of 2003) "overwhelming evidence in epidemiologic and medical articles, based on extensive research in various countries, conclude[es] that there is no valid proof that childhood vaccinations cause [T1DM]," adding that the expert stood alone in proposing the contrary – and that his stance reduced his credibility (while also being unreliable scientifically). *Id.* at *33, 34.

Petitioner attempts to distinguish *Baker*, arguing that the petitioner therein received the T1DM diagnosis much longer after vaccination, and that Dr. Classen's opinion was properly rejected for a number of reasons not relevant herein. Br. at 14. Nevertheless, *Baker* shows recognition that the injury at issue – diabetes – is not one likely to be acute in nature, making it unlikely to be suddenly triggered by the effects of vaccination. And worse for the present case – *as of over 20 years ago*, overwhelming epidemiologic evidence suggested vaccines do not cause diabetes.

The next decision rejecting a vaccine-diabetes association came three years later, *Meyers*, and it involved the DTaP vaccine (the form of Tdap almost exclusively administered to infants). *Meyers,* 2006 WL 1593947, at *1. The *Meyers* petitioner, as in *Baker,* also sought to establish that a child developed T1DM (and/or experienced later worsening of it) due to a series of DTaP vaccines he received early in his life. But the special master took note of *Baker's* treatment of the causal theory, which was dependent on the same reasoning (and even the same research and arguments of Dr. Classen), noting that the evidence offered for the theory was scientifically unreliable – and had been soundly rejected by the medical community, via other more persuasive

and reliable research. *Id.* at *5-7. In so doing, the special master also highlighted that evidence offered to support the theory was not specific to the DTaP vaccine. *Id.* at *7.

Petitioner also endeavors to argue why *Meyers* is not relevant herein, primary relying on factual distinctions between the cases. Here, she emphasizes, the claim was one of significant aggravation, with onset beginning after a series of Tdap vaccines. Br. at 14. She also points out that the petitioner in *Meyers* again relied on Dr. Classen's rejected theories. *Id.* However, she makes no mention of the other evidence, in the form of epidemiologic studies, that generally suggest vaccines do not – *cannot* – cause diabetes.

With the passage of another three years, a new decision was issued – again discrediting the contention that vaccinations could cause T1DM. *Hennessey*, 2009 WL 1709053. This time, the focus was the Hep B vaccine (as well as the slightly-distinguishable contention that the T1DM at issue had been aggravated rather than directly caused by vaccination), although again, the injured party was a child (a non-infant this time, but closer in age to B.B.) who had received vaccines as part of routine wellness care. *Id.* at *6. The special master deciding the case made note of the fact that the claim was part of an omnibus series involving diabetes as the injury. *Id.* at *5-6. The *Hennessey* petitioner again invoked the rejected Dr. Classen contentions and articles. *Id.* at *29-30. But this was not his exclusive support offered for the theory; he utilized a different expert as well, who maintained that vaccines could trigger the "autoimmune destruction of β islet cells," resulting in T1DM (or here, worsening it). *Id.* at 53. The special master deemed the theory scientifically unreliable, and this determination was affirmed both by the Court of Federal Claims and the Federal Circuit.

*Hennessey*, Petitioner argues, involved a different vaccine as well as a significant aggravation claim, and is otherwise "non precedential"[17] – but in fact is actually supportive of this claim, in Petitioner's view. Br. at 9. The onset was extremely short in comparison to this case, and the *Hennessey* expert relied on a theory of autoimmune causation that was overbroad, while Respondent's expert accurately explained the process leading to T1DM, along with the fact the disease can be mediated by direct viral attack, as well as usually featuring a latency that is lengthy. *Id.* at 10, 11. But B.B.'s onset was not too short, contrasted with his negative test results at the time of vaccination, and otherwise suggestive of a process that had not been latent but could only have begun suddenly, in the post-vaccination timeframe. *Id.* at 11-12. Thus, Petitioner reasons, *Hennessey* is not illustrative of how a "typical" diabetes injury case is addressed in the Program.

Petitioner correctly highlights some fact distinctions between *Hennessey* and the present case. But (besides simply listing diagnostic factors relevant to B.B.'s T1DM assessment that are

[17] Of course, other than Circuit decisions, essentially *all* Vaccine Program decisions are nonprecedential (with the exception of decisions issued by a judge of the Court of Federal Claims *specifically* in the context of a Program case that has been appealed *to that judge* via a motion for review). But this does not mean that comparable cases do not provide helpful guidance – especially when a theory echoes what has been rejected time and again in the past.

not in contention), her argument amounts to claiming that vaccine causation *has* to be considered a possibility, merely because of the somewhat short timeframe at issue between vaccination and onset. This reflects the kind of *post hoc ergo propter hoc* reasoning firmly rejected by controlling Circuit precedent. *U.S. Steel Group v. U.S.*, 96 F.3d 1352, 1358 (Fed.Cir.1996) ("But to claim that the temporal link between these events proves that they are causally related is simply to repeat the ancient fallacy: *post hoc ergo propter hoc*."). And in any event, and as discussed below, Petitioner has not offered reason to accept the theory that any vaccine could suddenly trigger T1DM – or that anything about B.B.'s particular presentation makes that more likely.

Yet another in the series of decisions rejecting the contention that vaccines cause T1DM – and perhaps the best prior relevant decision – is *Crutchfield*. That petitioner was an adult female, and she had received an MMR vaccine that she alleged resulted in her development of diabetes several months later. *Crutchfield*, 2014 WL 1665227, at *4-5. The *Crutchfield* claimant utilized the same immunologic expert as in *Hennessey*, offering the theory that an autoimmune process (with molecular mimicry as its mechanism) resulted in the destruction or attack on islet cells. *Id.* at *9. But the special master made a number of determinations based on the evidence that completely upended the theory: (a) the process by which the body destroys islet cells takes a long time, and occurs well before symptoms manifest, making it unlikely a vaccine could promulgate such a process in shorter timeframe, (b) relevant epidemiologic evidence strongly undermined any vaccine-T1DM association (supported by evaluations of the data from the Institute of Medicine), (c) Petitioner's expert's theory was on its own terms poorly articulated and facially unpersuasive. *Id.* at *10-18. Thus, causation was not demonstrated. *Id.* at *21. Petitioner makes no mention of *Crutchfield* in attempting to evade dismissal of this claim.

Finally, I also decided a claim involving T1DM as the alleged injury, although one caused by the HPV vaccine. *E.S.*, 2020 WL 9076620. That petition involved an 18-year-old who alleged a panoply of vaccine-caused conditions, including exacerbation of well-controlled T1DM after vaccination. *Id.* at *2-4. I found this aspect of the claim deficient (along with the entirety of the claim). *Id.* at *47-49. In so doing, I took note of the aforementioned well-reasoned decisions, all of which firmly and persuasively rejected the existence of any vaccine-T1DM association. *Id.* at *47. But I also determined that the expert opinion offered made unfounded and unreliable contentions about the HPV vaccine. *Id.* at *48. Petitioner argues *H.S.* involved a different vaccine, and also involved a significant aggravation claim (and otherwise addressed, as noted above, some of the prior cases referenced in *E.S.*).

None of these decisions preclude outright the claim that has been asserted, but they are well-reasoned and worthy of consideration – and all demonstrate the significant weaknesses of the argument that the biological processes that likely cause T1DM could be set into motion, or aggravated, by vaccination. All rely upon what is known about T1DM's likely pathogenesis and its lack of demonstrated vaccine association. These decisions show that it is unlikely that the

immune-stimulative effects of vaccination can produce this injury (regardless of the precise mechanism at issue). Moreover (and as well explained in *Crutchfield*), ample reliable epidemiologic evidence undercuts the conclusion that childhood vaccines could initiate T1DM. *See generally Crutchfield*, 2014 WL 1665227, at *15. Such evidence suggests causation is unlikely *regardless* of the theory presented or relevant vaccine.

## II. Petitioner's Causation Theory Is Unreliable and Unsupported With Preponderant Evidence

Admittedly, the theory offered herein is not precisely congruent with what prior petitioners have (unsuccessfully) proposed. (This is why I have gone to the trouble to issue a reasoned decision, rather than simply following the above-referenced decisions that revealed the utter absence of a vaccine-T1DM association.) Thus, although Dr. Axelrod relies upon the oft-rejected contention that molecular mimicry drives T1DM's alleged post-vaccination pathogenesis, he also incorporates into his theory the notion that cytokines produced in initial reaction to vaccination (and thus via the innate arm of the immune response) would become pathogenic due to the subsequent adaptive response (thereby proposing a theory that could apply to *any* vaccination event – touching all aspects of the immune process, in fact). Axelrod Rep. at 9. This particular amalgamation of disparate causation elements has not been expressly considered by a special master in the previously-reviewed T1DM cases.

Nevertheless, this causal theory is no more persuasive or credible than what was rejected in the older cases – and the fundamental weaknesses with arguments that vaccines can cause T1DM remain, so many years since the concept was first raised in a Vaccine Program case. (Indeed, as Dr. Willi observed, the very idea that an infection might lead to an autoimmune process resulting in T1DM was proposed in Gamble *over 50 years ago* – and yet that hypothesis remains unconfirmed by any reliable scientific or medical research. Willi Rep. at 5). The theory *itself* is not even all that "new." The causal mechanisms it relies upon are ones I routinely encounter in other vaccine injury cases, but just as often deem weak and/or inadequately supported by preponderant proof.

There are two foundational deficiencies with the theory that any vaccine could cause T1DM, and they were well-explained in *Crutchfield* ten years ago. First, the timeframe in which T1DM is understood to develop and then manifest with the classic indicia of diabetes is *far longer* than a month or less (as would be the case here, since B.B.'s symptoms allegedly began approximately within two weeks of vaccination). *Crutchfield*, 2014 WL 1665227, at *10. Dr. Willi credibly opined that T1DM is simply not an acute disease process, and the fundamental nature of the condition, or medical science's understanding of it, has not changed since the Program first rejected the notion that it could constitute a vaccine injury.

Petitioner's timeframe theory stands in stark contrast to what is known about T1DM's prolonged development. Petitioner notes that on the day of B.B.'s vaccination, May 14, 2021, B.B.'s urine testing revealed no glucose or ketones. Ex. 1 at 135. On June 26, 2021, 43 days later, B.B. was in severe diabetic ketoacidosis. *Id.* at 38. Petitioner argues that "this is not indicative of a slow process," implying that B.B. did not have diabetes on the day of vaccination and developed the disease rapidly – within 43 days following vaccination (or two weeks, if measured by the onset of B.B.'s symptoms). Br. at 11. But it is well-established that T1DM takes several months, if not years, to develop. Accordingly, this theory of rapid progression due to vaccination is untenable.

Second, there is substantial, reliable epidemiologic evidence undercutting the contention that *any* childhood vaccine could be causal of T1DM. *Crutchfield*, 2014 WL 1665227, at *13. Some items of this kind of evidence have also been filed in this case. *See* the IOM Report; Beyerlein; Morgan; Hviid; I.B. Hirsch et al., *Pathogenesis of Type 1 Diabetes Mellitus*, UpToDate 1, 40 (2023), filed as Ex. D, Tab 2 (ECF No. 25-34). This evidence unquestionably bears on causation – and I may legitimately consider it in weighing the proof before me. For even though claimants are not *required* by *Althen* to offer epidemiologic studies to support their claim, reliable studies that exist are relevant to a claimant's success – and where they do not support causation, they can play a role in defeating entitlement. *See Andreu*, 569 F.3d at 1379. Tellingly, Petitioner evades mention of this literature in arguing against the claim's dismissal.

In addition, to the extent Dr. Axelrod's theory differs from what prior special masters considered when ruling on T1DM claims, it nevertheless recycles contentions that are routinely rejected as medically and scientifically unreliable. For example, Petitioner seeks to invoke the intended/anticipated effect of vaccination at its outset, in which proinflammatory cytokines are produced. Indeed, this is why many vaccinated individuals feel "malaise" in the wake of a vaccination. *See, e.g.*, *Bielak v. Sec'y of Health & Hum. Servs.*, No. 18-761V, 2023 WL 35509, at *37 (Fed. Cl. Jan. 3, 2023) ("general post-vaccination malaise is not unexpected"). But this does not also mean that this vaccine-associated cytokine production is likely *pathogenic* – or even that the amount of cytokines produced could become so, especially in comparison to a wild infection (which is not self-limiting in the way a vaccine is). *See Palattao v. Sec'y of Health & Hum. Servs.*, No. 13-591V, 2019 WL 989380, at *37 (Fed. Cl. Spec. Mstr. Feb. 4, 2019). I therefore have often found unpersuasive and unreliable this kind of reasoning, which seeks to recast the intended or expected results of vaccination into the start of a pathologic process. *See e.g.*, *Palattao*, 2019 WL 989380, at *37 ("But claimants cannot transmute scientific evidence exploring how vaccines normally function in the immune system into a reliable and persuasive causation theory that any vaccine can be pathogenic without a more specific showing that applies to the circumstances at hand.").

Indeed, in service of these arguments Dr. Axelrod's report references specific items of literature I have been repeatedly confronted with in prior cases. For example, he cites Kashiwagi

for the proposition that vaccines rapidly cause increased cytokine production. But I have noted in other decisions that Kashiwagi simply does not establish that (a) the *levels* of cytokines produced would be sufficient to be pathologic, or (b) proinflammatory cytokines are initially *integral* to causing the harm leading to whatever disease process is alleged to have been vaccine caused. *See, e.g., Hayward v. Sec'y of Health & Hum. Servs.*, No. 15-005V, 2018 WL 2772495, at *17 (Fed. Cl. Spec. Mstr. May 4, 2018) (referencing a number of other special master decisions casting doubt on the probative value of Kashiwagi); *Dean v. Sec'y of Health & Hum. Servs.*, No. 13-808V, 2017 WL 2926605, at *17 (Fed. Cl. Spec. Mstr. June 9, 2017). The citation of Kashiwagi thus is nothing more than an unsuccessful effort to turn a study that provides a reliable *observation* about what vaccines generally do into proof that this kind of immune stimulation is *pathologic*.

This matter also (like so many others before it) seeks to shoehorn the autoimmune mechanism of molecular mimicry into a context in which the mechanism does not work. Thus, and as I noted above in my summary of Dr. Axelrod's expert report, Petitioner purports to establish amino acid sequence homology between aspects of the Tdap vaccine and a variety of protein components relevant to pancreatic insulin production. Axelrod Rep. at 7-8. Of course, this showing assumes cross-reaction would occur due to antibodies produced in reaction to the vaccine. But it absolutely has not been demonstrated that the vaccine (or its wild virus components) likely leads to the production of these attacking autoantibodies, let alone that they would drive the *initial* process leading to T1DM. Yet again, a Program claimant hopes to "wave the flag" of molecular mimicry and prevail – without grounding it in the true particulars of this matter.

Petitioner's theory therefore adds up to the following: (a) vaccines can rapidly upregulate proinflammatory cytokines, (b) cytokines may play a role in islet cell destruction (although it is not demonstrated this is an initiating role), (c) molecular mimicry is a pathologic mechanism relevant to some autoimmune disease processes, and (d) Dr. Axelrod was able to show some degree of homology between certain vaccine components and proteins relevant to the insulin-producing cells. In addition, the timing of these processes (fast cytokine production and then a period of two weeks or so for the adaptive immune production of autoantibodies) "fits" what B.B. actually experienced.

The gaps in this chain of medical and scientific suppositions are fairly evident, however. Even if vaccines can upregulate cytokines, *at what point* do cytokine levels become pathogenic, encouraging harmful, chronic inflammation? How does the theory "go" from focusing on cytokine upregulation to an adaptive immune response leading to the production of cross-reactive autoantibodies? Even where some amino acid sequential homology can be shown (by reference to protein peptide databases) between peptide sequences found in vaccine viral proteins and components of the islet cells responsible for insulin production, *how* is it likely the vaccine causes production of autoimmune antibodies that would drive this destruction of the insulin-producing cells? And why does all of the above deserve more evidentiary weight than unrebutted, large-scale

epidemiologic studies suggesting vaccines *do not likely cause T1DM*? Insufficient reliable evidence has been filed in this case to fill these holes. The theory implicates the vaccines at issue as pathologically affecting the immune response *in its entirety*, and based solely on Dr. Axelrod's flimsy say-so.

The idea of cytokines as driving pathology in T1DM is even rebutted by recently-published literature filed in this case. Respondent specifically offers Moran and Greenbaum for this purpose – studies that specifically evaluated if *blocking* the cytokines would impact T1DM's progression (specifically by reducing β cell loss), but did not make such a finding. *See, e.g.*, Greenbaum at 7 (characterizing the study's findings as "an unambiguous, but disappointing, clinical result"). Dr. Willi was in fact one of Greenbaum's co-authors. *Id.* at 1. Dr. Axelrod, by contrast, could offer nothing so directly on point to defend his general and speculative theory.

Petitioner's theory also assumes the vaccine reaction would be congruent with the immune response to a live, wild virus or bacterial infection. In fact (and although evidence that a wild virus or bacterial analog to a vaccine component is associated with a particular injury is relevant to the causation question), infectious responses are far more damaging, impacting the immune system by orders of magnitude greater than what a vaccination can accomplish. *See Martin v. Sec'y of Health & Hum. Servs.*, No. 17-250V, 2020 WL 4815840, at *27 (Fed. Cl. Spec. Mstr. July 17, 2020) ("There is a vast difference between the transient increase in cytokines that vaccination is intended to trigger (since an innate response is required for the vaccine to have immunogenicity) and the kind of harmful, ongoing inflammatory process that a wild infection causes."). As a result, the fact that *some* infections are thought to *possibly* encourage T1DM's progression does not make it appreciably more likely in this case that *vaccination* would – especially since the wild virus/bacterial analogs for the Tdap vaccine's primary components have not (unlike enterovirus) even been shown to be associated with T1DM. And as noted by Dr. Willi, speculation about an infectious driver of T1DM has not resulted in scientific confirmation, even 50 years after the possibility was raised. Willi Rep. at 5.

Given the above, Dr. Axelrod's overall theory is scientifically unreliable and does not reflect new scientific/medical thinking on a putative vaccine-T1DM association that has never been shown to exist. While it may not have been invoked before in this specific manner in any of the prior T1DM cases, it repeats the kind of rejected arguments made in many Vaccine Act cases about the impact of cytokines, how a vaccine might mimic the impact of a viral infection, and/or the ways stimulation of the immune system can purportedly become pathologic. And it gains no heft from the fact that Dr. Axelrod personally espouses it as valid – especially since he has not been shown to possess prior expertise in researching T1DM or its theoretical causal etiology. It is well established that a special master need not accept "expert opinion testimony that is connected to the existing data or methodology 'only by the *ipse dixit* of the expert,' or where 'there is simply too great an analytical gap between the data and the opinion proffered.'" *Jarvis v. Sec'y of Health*

*& Human Servs.*, 99 Fed. Cl. at 61 (quoting *Cedillo*, 617 F.3d at 1339). Respondent's experts, by contrast, deftly and succinctly established the weaknesses of Petitioner's theory. Dr. Willi in particular was far more credentialed in the subject and injury in question. His view warrants significant weight, and he was much more persuasive in explaining the deficiencies with Dr. Axelrod's theory.

Petitioner also attempts to differentiate this case factually from all the aforementioned prior Vaccine Program determinations but fails to persuasively do so. For example, she deems significant the fact that B.B. received more than one dose of the Tdap vaccine within a year, along with the meningococcal vaccine, reasoning that this was somehow unusual, and/or created immune stress on B.B. But these circumstances have not been shown to be medically or scientifically meaningful. Nothing has been offered in this case establishing that receipt of two vaccines at once makes T1DM more likely, and no reliable independent evidence was offered to show any medical community concerns about the dual impact of two vaccines in causing T1DM, or the receipt of two Tdap doses in less than a year. (In fact, it should also be noted that B.B.'s vaccination records reveal he received *eight Tdap doses* between May 2009 and May 2021 – yet no explanation is provided for why the prior doses did not trigger T1DM earlier, assuming his susceptibility). Br. at 2.

Petitioner's other factual arguments about why this claim is distinguishable from prior matters are similarly unavailing. While many prior Program cases rejecting causation in the context of T1DM did not involve the Tdap or meningococcal vaccines, nothing has been offered suggesting these two vaccines are *more likely causal* of T1DM – especially given the epidemiologic evidence. *See* Beyerlein; Morgan. The fact of B.B.'s "sudden" onset is also a red herring. It is a foundational legal principle in the Vaccine Program that *not all post-vaccination injuries are vaccine caused*. *See U.S. Steel Group*, 96 F. 3d at 1358. While a short timeframe from vaccination to onset can be meaningful in some instances, the speed at which a disease manifests does not constitute a *per se* factor favoring causation.[18] (It is in fact likely that B.B.'s T1DM process began *long before* vaccination, and only coincidentally manifested after).

I reiterate one particular overarching point. Despite due opportunity to locate and file new research or scientific/medical literature regarding T1DM that would even *indirectly* support a vaccine causal association, Petitioner has failed to do so. There are no new identified findings in the field that make it more likely as of 2024 that *any* vaccine can cause T1DM. No recently-filed studies specific to T1DM's etiology have been offered. And if such evidence existed, it is highly likely Dr. Willi – whose experience and competence in understanding T1DM far outpaced Dr.

[18] Short onsets can often be fatal to a claim. A claimant alleging the flu vaccine caused Guillain-Barré syndrome, for example, must show onset occurred within 3-42 days of vaccination – meaning in most circumstances an onset very close in time to vaccination defeats entitlement. *See* 42 C.F.R. § 100.3. But under Petitioner's logic, those circumstances would make causation more likely.

Axelrod's – would have been aware of it and addressed it. (At the same time, other newly-published research specific to T1DM, like Greenbaum, rebuts aspects of Petitioner's theory). As a result, there is no reason to give this claim more consideration.

## III. Petitioner's Process Objections are Spurious

Petitioner has raised a number of process-related objections to my determination to dispose of the claim at an early stage, based on the existing record and without additional expert inputs. None are persuasive or valid.

She suggests, for example, that it is legal error for a special master to create a "bright line" legal rule about whether T1DM as a putative vaccine injury should be considered at all, despite the Program's repeated rejection of theories that vaccines cause diabetes (with her counsel adding that she lacks the scientific expertise to evaluate the question).[19] Br. at 20.

But I did not dismiss the claim *ab initio*.[20] Rather, I accurately informed the Petitioner that the injury alleged has resulted in *no* favorable reasoned decisions in the entirety of the Program's life – strongly suggesting to me that this claim too would fail, since other special masters have looked carefully at the nature of diabetes but found it unlikely a vaccine could bring it into existence. But (allowing for the slim possibility that new science researching the subject might exist, and prove more favorable to such a claim), *I proposed briefing on the subject,* and expressly invited Petitioner to address my concerns with proof and legal argument. Order to Show Cause, dated February 27, 2024 (ECF No. 26). In this decision, I evaluate Petitioner's success in meeting this challenge, based upon the expert reports that were filed in this matter by both sides. As this lengthy decision reveals, I have given the argument the airing Petitioner claims to have been denied.

It also is neither unjust nor improper for a special master to take note of the Program's treatment of certain classes of claims when evaluating a newly-filed petition that appears to cover the same ground. Special masters, in fact, are *called* to do so – not to bury their heads in the sand simply because a claim involves a new petitioner, but rather to draw upon their hard-won expertise, as well as that of their predecessors, when deciding vaccine injury claims. *See Doe v. Sec'y of Health & Hum. Servs.*, 76 Fed. Cl. 328, 338-39 (2007) ("One reason that proceedings are more

[19] I find this particular objection especially disingenuous. Counsel in this case has represented Program petitioners for 26 years, and has generated hundreds of thousands of dollars in fees as a result. Moreover, this case was *initiated* with an expert report – meaning counsel took the time before filing the Petition to seek out the very medical/scientific expertise she disclaims the ability to assess. She had ample opportunity to evaluate the claim's likely merits, and to take into account that this kind of injury had never produced a favorable entitlement decision.

[20] Indeed, I would *never* do so, unless it was self-evident that some obvious jurisdictional issue, or essential claim element (for example, proof of receipt of a covered vaccine), was absent.

expeditious in the hands of special masters is that the special masters have the expertise and experience to know the type of information that is most probative of a claim."). This often can, and does, inure to a claimant's benefit – I would not, for example, force a petitioner to offer an expert opinion on whether the flu vaccine "can cause" Guillain-Barré syndrome, and in many cases I urge settlement of claims that I know are often decided in petitioners' favor.

The opposite also occurs, however. The Program has existed for approximately 40 years, and special masters have had the occasion to consider a myriad number of claims – including claims that are repeatedly asserted. Should special masters simply ignore prior adverse decisions? Or reference them only in an oblique manner, since the present claim is inherently "different"? And what to do when such prior relevant decisions stand as convincing determinations that a theory is unlikely to succeed? Petitioner counsels judicial inefficiency in favor of adopting the pretense that every newly-filed claim presents a chance of success – even where none has ever before so succeeded, and regardless of an absence of new scientific or medical discoveries pertinent to the theory.

Another unconvincing argument offered against dismissal is the contention that I am denying Petitioner a "full and fair" chance to prove her claim unless she is permitted additional expert discovery (presumably in addition to what she filed at the claim's outset). Br. at 21. Admittedly, even when a special master may have reasoned doubts a claim is likely to succeed, there are circumstances where expert input is still warranted – for example, on a topic that the special masters generally do not agree upon as to causation. *See, e.g.*, *Stewart-Robinson v. Sec'y of Health & Hum. Servs.*, No. 22-477 V, 2024 WL 4511807, at *8 (Fed. Cl. 2024) (allowing expert report on topic where special masters had reached varying conclusions).

At the same time, however, the Act and Vaccine Rules imbue special masters with considerable discretion to determine what discovery is merited in a given case, as well as how best to resolve the matter overall. *Burns v. Sec'y of Health and Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993); Vaccine Rule 3(b)(1). A special master must "endeavor[ ] to make the proceedings expeditious, flexible, and less adversarial, while at the same time affording each party a full and fair opportunity to present its case and creating a record sufficient to allow review of the special master's decision." Vaccine Rule 3(b)(2). The Vaccine Rules further direct the special master to "determine the format for taking evidence and hearing argument based on the specific circumstances of each case and after consultation with the parties." Vaccine Rule 8(a). Here, and as addressed at length above, I have explained carefully why I deemed this case properly dismissed at this stage, without further expert input. Allowing even more expert reports in a case as facially deficient as this one would only result in unneeded expense, without making Petitioner more likely to prevail.

Further expert input is especially unwarranted herein, since it is indisputable that Petitioner *initiated* this claim with an expert report – Dr. Axelrod's opinion.[21] Am I compelled to permit Petitioner to re-articulate her theory further, with even more expert input, in light of my reasoned objections, which arise in part from case law that existed *when* this claim was filed? How many iterations of supplemental or additional expert opinions should be permitted? And when would such an expert process end – when the claimant *agrees* the matter is ready for resolution, or otherwise "cries uncle"?[22] This is not a determination entrusted to petitioners – and since my denial of additional expert discovery is based on my reasoned view that Petitioner's theory has been "fully and fairly" evaluated, the natural desire of any petitioner to keep a claim going, revising it each time a special master notes a deficiency, should not carry the day.

Finally, Petitioner contends that the rarity of vaccine injuries should inform how any petition is received. *See, e.g.*, Br. at 7 ("even though much of the literature discusses how, in general, these vaccines do not cause T1DM, they do not say that *they cannot in rare case*[s]" (emphasis added)). Because "this Program is for rare events," I must stand aside and let the evidence flow in, for "this is why we have this Vaccine Program." *Id.*

This contention has, however, been repeatedly rejected by the Circuit, which emphasizes a claimant's foundational *evidentiary obligation* to offer preponderant evidence in support of a theory. *See Hibbard v. Sec'y of Health & Hum. Servs.*, 698 F.3d 1355, 1366 (Fed. Cir. 2012) (emphasizing that a petitioner must satisfy the preponderant standard in order to obtain an award for compensation). When this cannot be done, it does not matter that it always remains possible the vaccine was causal. The Program does not operate as an "insurance policy," in which every post-vaccination injury is compensated on account of rarity. If the preponderant standard of proof is not met, the claim is properly dismissed, despite a lingering possibility that causation *might* still have occurred.

[21] Program petitioners should usually abstain from obtaining an expert's assistance before a claim is filed (beyond maybe a short consultation). Special masters are ultimately responsible for paying fees and costs in a case, and a claimant risks denial of reimbursement for an expert's potentially-expensive time if such assistance is obtained without first gauging the special master's views regarding the need for or contours of such expert input. Here, had Petitioner waited, I would have warned her against filing a report unless new scientific/medical evidence could be identified making it more likely that vaccines can cause T1DM; the anticipatory report filed by Dr. Axelrod failed to accomplish this (underscoring the absence of such evidence generally).

[22] In fact, it is my experience that Program petitioners, left to their own devices (and well aware of the Program's infinitely-generous fee shifting statute, shielding them from personal financial liability for expert reports), would in many cases happily file *round after round* of reports, modifying the theory each time a special master questions its persuasiveness or reliability. Doing so rarely brings clarity to a dispute, and more often than not devolves into a tit-for-tat battle between the experts that could go on *ad infinitum*, unless the special master exercises needed control over the matter. *See e.g.*, *D.G. v. Sec'y of Health & Hum. Servs.*, 2019 WL 2511769, at *186-89 (Special Master Millman criticizing expert's ever-changing opinion, displayed in his 10 expert reports, and stating that "an expert who can never made up his mind is practically useless").

I otherwise give little weight to arguments that rely on rarity of injury to evade basic evidentiary requirements.[23] As former Special Master Hastings in *Crutchfield* aptly noted (in the context of addressing the utility of epidemiologic studies):

> It is, in fact, *always* true that epidemiological studies can *never* prove definitively that Factor A *never* caused Condition B. Even when large studies fail to identify an association between Factor A and Condition B, it is *always theoretically possible* that Factor A causes Condition B in a very small number of cases, an effect too rare for the study to detect. But it is not Respondent's burden in this case to prove that it is *impossible* that the . . . vaccination can cause Type 1 diabetes. It is, rather, the *Petitioner's* burden to show not only that the . . .vaccine can cause Type 1 diabetes . . . but also that her own . . . vaccination did cause Petitioner's Type 1 diabetes.

*Crutchfield*, 2014 WL 1665227, at *15 (emphasis in original). Petitioners cannot hide behind rarity of injury arguments whenever the evidence comes up short.

Vaccine Program cases are not parlor games in which attorneys dream up clever causation arguments to evade obvious causation dead-ends. Rather, claimants and their counsel should advance only causal theories supported by *reliable scientific evidence specific to the vaccine in question*. It is also reasonable to expect experienced counsel to consider whether a kind of claim has met with success in the past – and whether they can muster new evidence warranting reconsideration of a particular kind of alleged injury. Where this does not occur, a special master acts properly in dismissing the claim, rather than engaging in further wasteful judicial process that distracts from the adjudication of meritorious and reasonably-disputed petitions (of which there are many).

## IV. Claims Asserting T1DM as a Vaccine Injury Lack Reasonable Basis

Because it is preponderantly evident (based on existing medical science) that the vaccines at issue cannot cause T1DM, and because Petitioner has identified no new scientific/medical thinking on the subject, this specific matter lacks reasonable basis *going forward.* (Arguably, the matter also lacked reasonable basis *as of its filing*. But the bar for reasonable basis is extremely low, and since the theory offered was not *wholly* identical to what has been rejected in this context

[23] Rarity is already "priced into" the Program in several ways. For example, claimants can prevail *without* direct proof of causation, and thus are not compelled to offer evidence like a solid epidemiologic study supporting a vaccine-injury association. The burden of proof is preponderance, moreover, rather than something higher. And it is never a defense to a vaccine injury claim to maintain that vaccines are generally "safe" for the public.

before, I will allow *some* fees for work *reasonably* performed to date).[24] I will not pay fees or costs for any *additional* work performed on this matter, however, including appeals.

Present counsel is admonished to avoid again advancing a claim alleging T1DM as a vaccine injury *unless* new scientific or medical evidence can be presented that would address the causal deficiencies so often identified in prior cases. Certainly, the evidence provided in this case does not pass that test. Other Program counsel are warned similarly to not bring this kind of claim in the future.

## CONCLUSION

I am certain that B.B. and his family have suffered in management of his T1DM, and I take no personal pleasure in having to dismiss the claim. But preponderant evidence does not support Petitioner's causation theory – and based on present science, *could* not. She is therefore not entitled to compensation.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[25]

**IT IS SO ORDERED.**

s/Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[24] I urge counsel to evaluate carefully what fees she requests, given her oft-observed proclivity for overbilling on Program matters. *See, e.g.*, *Iniguez v. Sec'y of Health & Hum. Servs.*, No. 18-1537V, 2023 WL 3729843, at *3 (Fed. Cl. Spec. Mstr. May 30, 2023) (finding "a chasm-wide gap" between the amount the case settled for, and the fees billed by Ms. Roquemore).

[25] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.